

AD-A113 043

INDIANA UNIV AT BLOOMINGTON SCHOOL OF MEDICINE
SLEEP DEPRIVATION AND EXERCISE TOLERANCE.(U)
JAN 82 B J MARTIN

F/6 6/19

DAMD17-81-C-1023

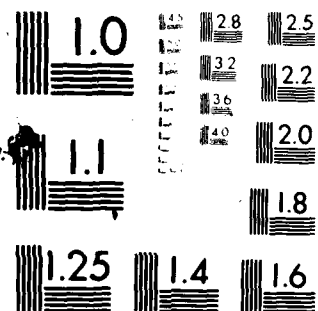
NL

UNCLASSIFIED

1 1 1
AD-A113 043



END
DATE
FILMED
5-82
DTIC



MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS-1963-A

(12)

UNCLASSIFIED

AD _____

REPORT NUMBER 1

Sleep Deprivation and Exercise Tolerance

Annual Summary Report

Bruce J. Martin, Ph.D.

January 1982

Supported by

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND

Fort Detrick, Frederick, Maryland 21701

Contract No. DAMD17-81-C-1023

Indiana University School of Medicine

Bloomington, Indiana 47405

Approved for public release; distribution unlimited

The findings in this report are not to be construed as
an official Department of the Army position unless so
designated by other authorized documents.

DTIC FILE COPY

DTIC
ELECTE
APR 5 1982
S H D

AD A11 3043

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
	AD A113 093	
4. TITLE (and Subtitle)		5. TYPE OF REPORT & PERIOD COVERED
Sleep deprivation and exercise tolerance		Annual: 2/1/81 to 1/31/82
		6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s)		8. CONTRACT OR GRANT NUMBER(s)
Bruce J. Martin		DAMD 17-81-C-1023
9. PERFORMING ORGANIZATION NAME AND ADDRESS		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
Indiana University School of Medicine Bloomington, IN 47405		62777A.3E162777A879.BF.084
11. CONTROLLING OFFICE NAME AND ADDRESS		12. REPORT DATE
U.S. Army Medical Research and Development Command Fort Detrick, Frederick, Maryland 21701		1/82
		13. NUMBER OF PAGES
		9
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)		15. SECURITY CLASS. (of this report)
		Unclassified
		15a. DECLASSIFICATION/DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report)		
Approved for public release; distribution unlimited		
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)		
18. SUPPLEMENTARY NOTES		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number)		
sleep; exercise; fatigue		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number)		
<p>The purpose of the study is to identify the effects of sleep deprivation on the ability of humans to tolerate standard forms of endurance exercise. Standard techniques in human exercise physiology are utilized.</p> <p>To date, the results indicate that acute sleep loss of 30 to 36 hours a) does not alter the maximal oxygen uptake, while it does reduce maximal</p> <p>(CONTINUED ON ADDITIONAL SHEET)</p>		

20. Abstract, Continued:

heart rate; b) leaves metabolic rate during exercise at a constant external work load unchanged, while heart rate is reduced and ratings of perceived exertion are elevated; c) reduces tolerance of prolonged heavy exercise at three-fourths of the maximal oxygen uptake by about 10%, with wide variation noted among individuals, and d) fails to significantly change work loads selected for equal effort during short-term heavy exercise.

We conclude that acute sleep loss of 30 to 36 hours has relatively minor deleterious effects on endurance exercise performance.

Accession For	
NTIS GRA&I	<input checked="" type="checkbox"/>
DTIC TAB	<input type="checkbox"/>
Unannounced	<input type="checkbox"/>
Justification	
By	
Distribution	
Available to	
Available and/or	
Dist	Special
A	



TITLE PAGE

AD _____

REPORT NUMBER 1

Sleep Deprivation and Exercise Tolerance

Annual Summary Report

Bruce J. Martin, Ph.D.

January 1982

Supported by

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND

Fort Detrick, Frederick, Maryland 21701

Contract No. DAMD17-81-C-1023

Indiana University School of Medicine

Bloomington, Indiana 47405

Approved for public release; distribution unlimited

The findings in this report are not to be construed as an official Department of the Army position unless so designated by other authorized documents.

SUMMARY

The purpose of the study is to identify the effects of sleep deprivation on the ability of humans to tolerate standard forms of endurance exercise. Standard techniques in human exercise physiology are utilized.

To date, the results indicate that acute sleep loss of 30 to 36 hours a) does not alter the maximal oxygen uptake, while it does reduce maximal heart rate; b) leaves metabolic rate during exercise at a constant external work load unchanged, while heart rate is reduced and ratings of perceived exertion are elevated; c) reduces tolerance of prolonged heavy exercise at three-fourths of the maximal oxygen uptake by about 10%, with wide variation noted among individuals, and d) fails to significantly change work loads selected for equal effort during short-term heavy exercise.

We conclude that acute sleep loss of 20 to 36 hours has relatively minor deleterious effects on endurance exercise performance.

FORWARD

For the protection of human subjects the investigator(s) have adhered to policies of applicable Federal Law 45CFR46.

BODY OF REPORT

STATEMENT OF THE PROBLEM

Sleep deprivation is a common occurrence in both the military and civilian spheres. In many instances, severe or prolonged exercise follows sleep loss. Despite this frequent occurrence, little is known of the effects that sleep deprivation may have on subsequent exercise.

BACKGROUND

Several previous studies have approached the question of the relationship between sleep loss and subsequent exercise. In some of these, sleep deprivation was combined with other stressors such as prior exercise or caloric restriction, rendering identification of the specific effects of sleep loss difficult (1, 2). Other studies have looked more specifically at exercise after an uncomplicated sleepless period, but have utilized either indirect (3) or inexact (4) measures of exercise responses or tolerance. This paucity of information contrasts with that available concerning other aspects of sleep deprivation and of the sleep deprivation - exercise relationship. For example, considerable work has been concerned with the psychological and physiological effects of sleep loss at rest (5, 6). In addition, many studies have examined the influence that heavy exercise may have on subsequent sleep (7).

It was in this context that our first study of sleep loss was made; it was partially complete at the time of application for the present Contract. We found that a 30 hour sleepless period had no influence on heart rate, metabolic rate, or ventilation, during cycle ergometer exercise at 25, 50, or 75% of the maximal oxygen uptake. The sleep loss period did increase ratings of perceived exertion during the heavier exercise levels. These perceptual changes were abolished after one night of unlimited sleep. We also found that the maximal oxygen uptake was unchanged by the sleep free period, while the maximal exercise heart rate was slightly but significantly reduced. This last finding contrasts with an earlier study showing a 4% fall in maximal aerobic power after sleep loss (8); in that study, the sleep loss period had been 72 hours, a much more severe stress and a possible explanation for the difference between studies.

APPROACH TO THE PROBLEM

These previous results suggested that the psychological effects of sleep deprivation could be detrimental to prolonged exercise performance. Studies were then performed that attempted to specify any performance decrement, and to correlate any changes in performance with changes in physiological or psychological responses to exercise.

RESULTS AND DISCUSSION

In the first year of this Contract, two studies have been performed which

will be described in turn.

First, tolerance of exercise at 75% of the maximal oxygen uptake was measured after a 36 hour sleepless period. Eight subjects participated, with the mean performance decrease about 10%, a statistically significant change. There was wide individual variation in this result: four of the eight subjects showed performance changes of less than 5%, while the four others had at least 15% decreases in work time. This performance decline came in the face of added incentives for work in the sleep deprived condition. Since subjects must be aware of their sleep status, we were concerned that they might expect to perform more poorly after sleep loss, with this expectation then producing its own self-fulfillment. To mitigate this effect, all external cues to work time were removed from the subjects, and they were simply told that they would receive payment for each minute worked on the treadmill, with the payment scale doubled after sleep loss. This design increases our confidence that a 36 hour sleepless period is sufficient to measurably worsen exercise performance.

Our attention then turned to the physiological mechanisms - or the psychological mechanisms - that could explain this performance decrement. The most obvious change induced by sleep loss in the physiological and psychological responses to prolonged exercise in the previously described study was a rise in ratings of perceived exertion. Because this rise was greatest in the four subjects showing the greatest decline in work tolerance, we hypothesized that this psychological effect of sleep loss might determine the performance decline. Physiological effects of sleep deprivation were less apparent: heart rate and metabolic rate were unchanged, while minute ventilation was elevated during the latter half of the exercise period.

We thus performed a study to investigate the psychological effects of sleep deprivation on subsequent exercise. We hypothesized that persons most influenced by sleep loss psychologically would most dramatically alter their perceptions of exercise intensity (and thus, potentially, exercise performance). Twenty-four persons were thus studied in order to search for a possible correlation between psychological changes and work load selection. In this study, we allowed subjects to choose their own work rate such that the perceived exertion was the same both in the control situation and after sleep deprivation. Standard psychological measures of sleepiness and mood were made before and after exercise; we hypothesized that persons most altered in mood (toward fatigue, tension, depression, and confusion) and in sleepiness would choose the most greatly underestimated work loads after sleep loss. To our surprise, we found that the mean work loads selected to provide equal perceived exertion were identical in the control and sleep loss conditions, and that there was no correlation between alterations in mood and work load choice. We did find, as others have in the past, that the sleep loss episode led to significantly elevated fatigue, confusion, depression, anxiety, and tension (9); but, these mood changes did not translate into changes in work load selection. We now feel that the previously measured increased perceived exertion after sleep loss may have been a spurious result, since it occurred under more biased circumstances than the present results that failed to confirm it. In addition, in this study we found a significant heart rate decrease during exercise at equal work loads and at equal metabolic rate.

CONCLUSIONS

Based on this first year of work, we conclude that exercise performance is decreased after sleep loss for as yet unknown reasons. Many aspects of the physiological response to exercise after sleeplessness remain to be measured; some of these are planned for the upcoming year.

RECOMMENDATIONS

Sleep loss of up to 36 hours is relatively ineffective as a direct inhibitor of exercise performance. For example, its effects are much less than that of prior exercise, or altered diet, and those imposed by relatively small changes in terrain, burdening, or environmental conditions. Two cautions must be mentioned here. First, little is known of the potential cumulative effects of more prolonged sleeplessness. Second, these studies have all examined exercise in the situation where it is required; if exercise were to be initiated by the subject in a self-motivated manner, it might be that the deleterious effects of sleep loss would be greater.

Literature Cited

1. Soule, R. and R. Goldman. Pacing of intermittent work during 31 hours. Med. Sci. Sports 5: 128-131, 1973.
2. Opstad, P., R. Ekanger, et al. Performance, mood, and clinical symptoms in men exposed to prolonged heavy physical exercise and sleep deprivation. Aviat. Space Environ. Med. 49: 1065-1073, 1978.
3. Holland, G. Effects of limited sleep deprivation on performance of selected motor tasks. Res. Quart. 39: 285-294, 1968.
4. Brodan, V., M. Vostechovsky, et al. Changes in mental and physical performance of sleep deprived healthy volunteers. Activ. Nerv. Sup. 11: 175-181, 1969.
5. Horne, J.A. A review of the biological effects of total sleep deprivation in man. Biol. Psychol. 7: 55-102, 1978.
6. Florica, V., E.A. Higgins, et al. Physiological responses of men during sleep deprivation. J. Appl. Physiol. 24: 167-176, 1968.
7. Walker, J.M., T.C. Floyd, et al. Effects of exercise on sleep. J. Appl. Physiol. 44: 945-951, 1978.
8. Harris, W., and J.F. O'Hanlon. A study of recovery functions in man. U.S. Army Technical Memorandum 10-72, Aberdeen Research and Development Center, MD, 1972.
9. Bonnet, M.H. Sleep, performance, and mood after the energy expenditure equivalent of 40 hours of sleep deprivation. Psychophysiology 17: 56-63, 1980.

PUBLICATIONS

Martin, B. J. Effect of sleep deprivation on tolerance of prolonged heavy exercise. Eur. J. Appl. Physiol. 47: 345-354, 1982.

Martin, B. J., and R. Haney. Self-selected exercise intensity is unchanged by sleep loss. Eur. J. Appl. Physiol. (In Press)

Both of these publications have been supported in part by this contract.

R U

DISTRIBUTION LIST

4 copies

USAMRDC (SGRD-RMS)
Fort Detrick
Frederick, MD 21701

12 copies

Defense Technical Information Center (DTIC)
ATTN: DTIC-DDA
Cameron Station
Alexandria, VA 22314

1 copy

Dean
School of Medicine
Uniformed Services University of the
Health Sciences
4301 Jones Bridge Road
Bethesda, MD 20014

1 copy

Commandant
Academy of Health Sciences, US Army
ATTN: AHS-CDM
Fort Sam Houston, TX 78234

**DAI
ILM**